

Cutaneous viral infection

Definition of viruses:

- Viruses are infectious agents; they don't possess ribosomes or other cellular organelles so depend on other cells for replication (obligate intracellular parasites).

Viruses are differentiated from other microorganisms by:

1. They don't grow on artificial media.
2. They don't divide by binary fission.
3. They contain either DNA or RNA, never both.
4. They contain no ribosomes or other cellular organelles.
5. They are not sensitive to antibacterial antibodies.

Pathogenesis of viral infection in skin:

Viral infection may occur in skin in 3 different ways:

1. Direct inoculation:

Direct inoculate and replicate in epidermis as wart, molluscum contagiosum.

2. Systemic infection:

Skin lesion produced by systemic viral infection called exanthemata which occur by viremia so the dermis infected earlier than epidermis.

Examples for viral exanthemata:

- Macular (rubella and infectious mononucleosis)
- Maculopapular (measles)
- Vesicular (chicken pox)

3. Local spread from internal focus:

As in HS and HZ: in which local spread of virus to skin after reactivation of latent viral infection present in peripheral nerves.

Host response to viral infection:

1. Non immunological:

Cells infected with virus produce interferon which diffuse to other cells and make them less vulnerable to the penetration by new viruses.

2. Immunological:

Ab

T-cell

Humoral immunity <i>recurrent</i>	Specific cell mediated immunity <i>by recurrent</i>
<ul style="list-style-type: none"> Represent major host defense against <u>reinfection</u> with the same virus but not important in <u>primary</u> viral infection. Mediated by antibodies by: <u>Ab</u> <ul style="list-style-type: none"> Neutralize virus by preventing its <u>attachment</u> to target cell. Enhancement of virus uptake by <u>phagocytic</u> cell. <u>Complement</u> mediated lysis of infected cell. 	<ul style="list-style-type: none"> Important in handling <u>primary</u> and <u>recurrent</u> viral infection. Mediated by <u>sensitized T-cell</u> by: <ul style="list-style-type: none"> Direct lysis of infected cell by <u>cytotoxic lymphocytes</u>. Liberation of <u>lymphokines</u> which attract <u>phagocytic</u> cells.

Skin disease caused by viruses

1) PICORNAVIRUS group: (very small viruses)

Enteroviruses

Poliomyelitis,
Coxsackie virus A & B

Rhinoviruses

Echoviruses

Common cold, foot, mouth disease

2) MYXOVIRUS group: (no Cut. manifestation in this group except in measles)

Orthomyxoviruses

Influenza virus

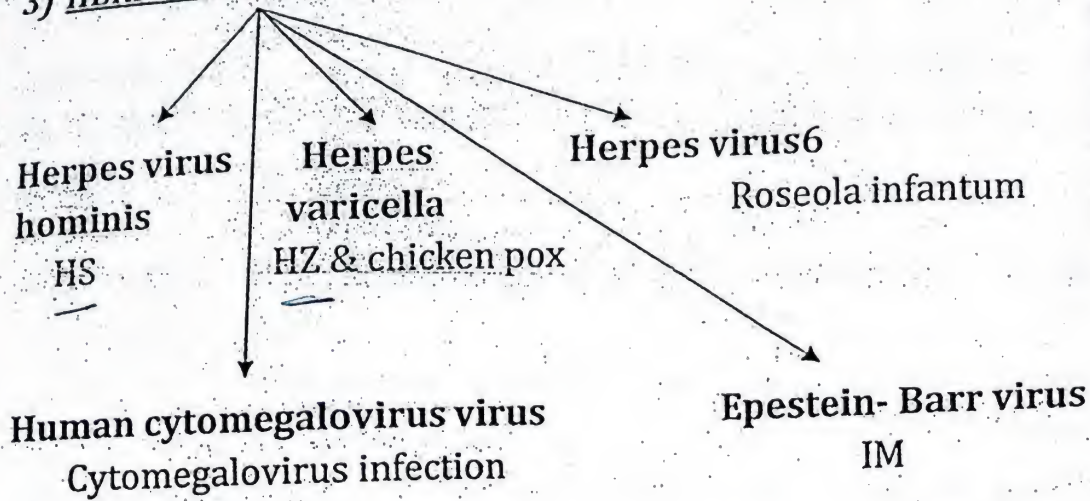
Paramyxoviruses

Paramyxoviruses
Parainfluenza, mumps

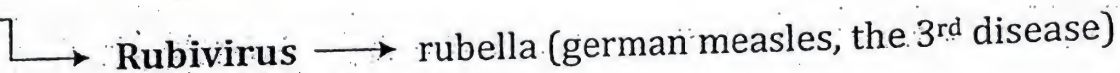
Morbillae virus
Measles

Pneumovirus
Respiratory
syncytial viral infect.

3) HERPES VIRUS group:



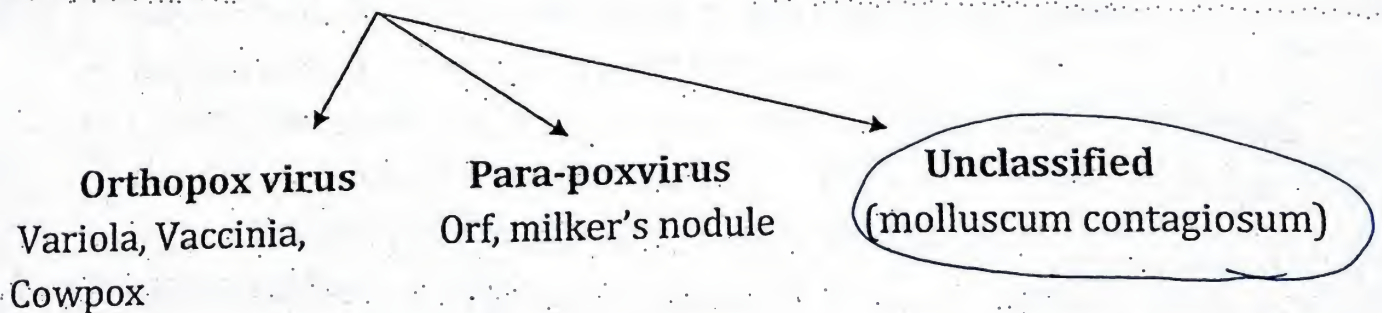
4) TOGAVIRUSES group:



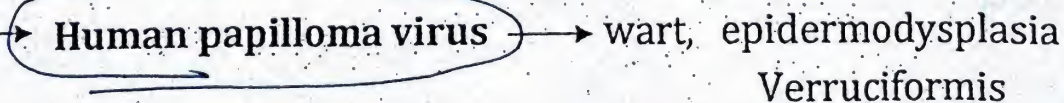
5) Human parvovirus B19:



6) POXIVIRUS group:



7) PAPPOVA virus group: (group of DNA potentially oncogenic viruses)



Measles

Def: measles is a high contagious acute viral disease characterized by high fever, cough, coryza, conjunctivitis and koplik's spot which followed by generalized macular and papular rash.

E/P: cutaneous eruption is the result of CMI response against measles virus.

1. Typical form:

- After IP of 10 days, start fever and malaise and associated with:
 - Upper respiratory catarrh. - Conjunctivitis, photophobia.
 - Koplik's spots: white spots with bright red areola on buccal mucosa opposite the premolar teeth and seen from the 2nd day.
- The cutaneous eruption develop on the 4th day starting on forehead and behind ears then spread to rest of face, trunk and limbs. The rash start macular, then soon become dull red papules which may be diffuse or coalesce in irregular pattern.
- Rash subside on the 6th or 10th day with residual brownish stain or fine desquamation.


2. Modified measles:

- IP may prolong to 14-20 days.
- Usually develop in children who have been immunized with IGs after exposure to the disease or who have been protected with immunization before the age of 15 months.
- Usually the prodromal stage may be absent or decreased to 1-2 days. Cough, conjunctivitis may be minimal or abscent. Koplik's spots are few or abscent and eruption are sparse and milder.

3. Atypical measles:

- This is the paradoxical form of measles; occur in children who exposed to natural measles with previously received vaccine.
- Manifested by high fever, cough, headache, myalgia, abdominal pain, pneumonitis, pleural effusion and unusual rash on hand and feet (macular, vesicular, peticheal)

Complication: encephalitis (most serious), bronchopneumonia, enteritis, otitis media are less seen than before.

DD: - other exanthems:  scarlet fever - drug eruption.
Rubella
roseola infantum
erythema infectiosum.

TTT:

- Human IGs given within 5 days of exposure may prevent or attenuate infection.
- Isoprinosine are said to modify the course of disease.

Rubella (german measles)

Def: common viral infection affects children and adults caused by rubivirus.

C/P: IP of about 18 days, the disease pass into 3 stages:

- **Prodromal stage:**

Not seen in children, only in adults. It lasts 1-5 days. Consist of fever, malaise, headache, conjunctivitis, sore throat and enlargement of cervical lymph gland also suboccipital and post auricular. Lymph node enlargement starts 5-7 days before rash and reach maximum in 1st or 2nd day of rash. Prodromal stage may have rash in form of dull red macules or petechiae in 20% of patients.

- **Cutaneous eruption:**

Start first on face then trunk and limbs, consist of pink macules which first are district then become confluent on face in form of diffuse erythema.

- **Stage of fading out:**

During 2nd day the face start to clear, macules disappear from face on 3rd day, then trunk start to clear. On 4th day; eruption on limbs disappears.

Diagnosis: the rash and the enlargement of suboccipital gland are characteristic.

Complication: if rubella occurs in the first 16 wks of pregnancy, 30% chance of fetal damage which is higher in the first month (50%-60%) and lower in the 4th month (5%). Damage includes:

(Cardiac abnormalities, deafness, eye lesions, CNS damage, thrombocytopenic purpura).

Prophylaxis: live attenuated rubella virus (vaccine) given to school children at age of 11-14yrs.

Erythema infectiosum

Def: Infectious disease caused by human parvovirus B19, seen in children 2-10 yrs old.

C/P:

- IP is 5-14 days, no prodromal stage.
- Disease start with red papules on face which, coalesce to form hot diffuse erythema giving the face (slapped appearance). Within the next 2-4 days facial erythema subside and maculopapular rash appear on proximal extremities then hands, feet, trunk. The rash of specific lace-like pattern.
- Eruption fade in 6-10 days and May recurre in previously affected sites for 2 wks.

Diagnosis: demonstration of IgM abs against parvovirus.

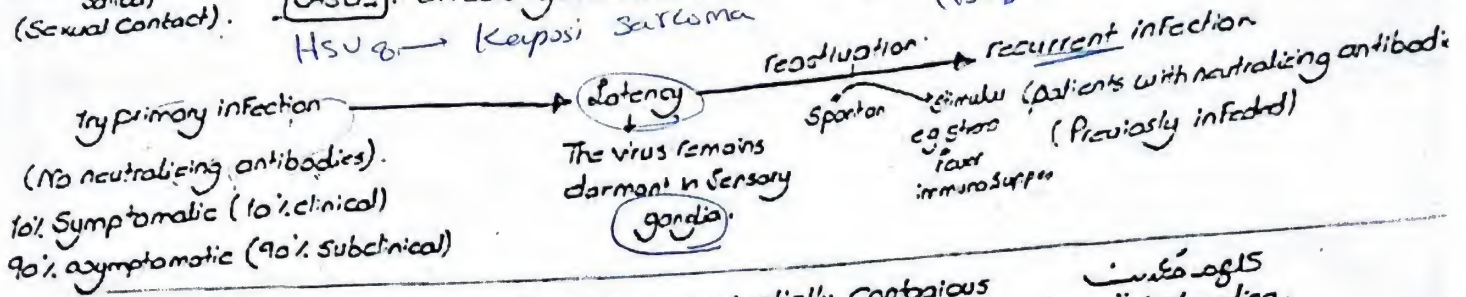
TTT: no specific ttt.

Viral Infections

① Herpes Simplex viruses

HSUs have worldwide distribution and produce primary, latent and recurrent infections.
There are ② subtypes of HSU which show no cross immunity:

① HSV-1 affects skin and oral mucosa (Herpes labialis)
② HSV-2 affects genital areas (HSV-1 may also be found in genital infection « Oro-genital contact with contaminated saliva)
HSV-8 → Kaposi Sarcoma
HSV-3 → chickenpox & shingles



Diagnosis → All persons infected with HSU are potentially contagious whether or not the lesions are visible because of asymptomatic viral shedding.

① Viral Culture

② Tzanck Smear

For rapid preliminary diagnosis
it shows multi-nucleated epithelial giant cells (fusion of infected keratinocytes)
It does not differentiate between HSV and VZV

③ DFA (Direct Fluorescent antibody) assay

→ able to distinguish between HSV and VZV.

④ PCR: Rapid / Sensitive / Specific Method to detect HSV DNA

⑤ Serological Tests (Western blot) ✓ gold standard for serological assay

99% Sensitive & Specific for HSV antibodies.

Other Tests That Can Differentiate between HSV-1 & HSV-2 antibodies
Depend on (Type-specific glycoprotein)
G1 From HSV-1 & G2 From HSV-2.

⑥ Biopsy

to exclude cutaneous conditions that can mimic HSU
eg → Vesicular eczematous dermatitis
or recurrent FDE on genitalia.

Histopathology:

(1) Ballooning degeneration of epidermal cells → marked acantholysis → intraepidermal vesicles
(2) Eosinophilic intranuclear bodies surrounded by a clear halo → are usually seen in balloon cells.

Clinical Manifestations of HSV-1 & HSV-2

ImmunoCompetent individual

Immuno-Compromised individual

HSV-1

Herpes labialis

→ primary infection usually asymptomatic
• Herpetic gingivostomatitis
• Herpes labialis
• Herpetic whitlow.

• widespread local infection
• Chronic ulcers
• Disseminated cutaneous infections.
• Disseminated visceral infections.

HSV-2

Herpes Genitalis

→ primary infection usually asymptomatic
• Herpes genitalis (primary & recurrent)

Herpes simplex

Def:

HS is one of the commonest viral infections. The disease has recurrent nature. The disease runs a mild course except in immunodeficiency and patient on cytotoxic drugs in which the course of disease is aggressive.

C/O:

- Herpes virus hominis type I: which affect skin and mucous membrane.
- Herpes virus hominis type II: affect genital areas.

Mode of infection: by direct inoculation, patients with recurrent HS may have the virus in tears or saliva.

vertical from mother → Eczema
touch
- sexual
- droplet

Primary infection:

a) Herpes virus hominis I: start mainly in infancy or early childhood (1-5yrs) where they are usually minimal and subclinical but severe cases of 1ry infection may occur in:

- Atopic dermatitis and other skin disease that may lead to wide spread cutaneous lesion (Kaposi's varicelliform eruption).
- Infant with congenital defect of immunity.
- Malnutrition
- Patient with malignancy of the RES.

b) Herpes virus hominis II: Infection is nearly always genital, and starts after puberty.

Recurrent infection: after 1ry infection, herpes virus remain latent in the sensory nerve ganglia or in neurons of peripheral nerves or in schwann's cells of sensory nerves which supply the 1ry infected area. Under certain conditions like exposure to cold, trauma, sun, certain foods, reactivation of latent virus occur to produce recurrent infection.

Primary Herpes Simplex

IP 3-8 days

- affects children (2-5 yrs).
- Prodrome:
 - Fever, Malaise
 - Localized burning
 - Tender lymphadenopathy
- occurs before the onset of lesions
- Lesions: Large painful vesicles on erythematous base (no tendency to grouping)
 - vesicles → pustules → erosions.
 - Crusting → Spontaneous resolution (2-6 weeks)
- Regional LNs are enlarged & tender.

Recurrent

- grouped small vesicles on erythematous base
- The vesicles are replaced by crusts
- Healing occurs within a week → normal skin

- Recurrent Condition.
- Regional LNs are not enlarged (unless any infection of vesicles).

- prodrome: The onset is usually preceded by a prodrome of localized itching & burning & erythema (only severe)

→ no fever or malaise.

Clinical Varieties:

- 1) Herpes Labialis (Facialis):
 - * Commonest
 - * Site: (Lips) are the most frequent site.
 - Nose - cheeks - ears → may also be affected.
 - * post-herpetic erythema Multiforme may occur (within 9 days) (Herpes-associated erythema Multiforme).

Eczema Herpeticum (Kaposi's Varicelliform eruption).

- * with HSV → in patients with pre-existing dermatoses e.g.
 - atopic dermatitis (AD)
 - Borium disease
 - MF, PF
 - ichthyosis, burn.

- Clinically:
 - Extensive eruption of vesicles & pustules occur mainly on areas of pre-existing dermatosis + Fever.
 - Face (usually severely affected) + marked edema.
 - The presence of Monotonous discrete 2-3 mm Hemorrhagic Crusts Diagnostic.

Keratoconjunctivitis

- Can be primary or recurrence
- recurrence → present with branching dendritic Corneal ulceration (seen with fluorescein stain)
 - Can lead to scarring & blindness.

gingivostomatitis (Very painful) # by systemic antiviral

Herpetic whitlow: HSV infection of the digits in dental & medical personnel (don't routinely use gloves)

Herpetic Folliculitis: of the bearded region in males

HSV pneumonia: (Fatal)

HSV encephalitis: Manifest as: Seizures / Irritability / tremors / bulging Fontanelle

HSV in compromised host (Disseminated HSV):

- Etiology:
- a) Wiskott Aldrich Syndrome
 - b) Lymphoma - Leukaemia
 - c) Severe burn
 - d) Prolonged immunosuppressive therapy
 - e) AIDS.

عنه
ايضاً
ممنوع
بالتامة

Manifestations

Chronic ulcerative HSV

- Persistent ulcers & erosions starting on the face or perianal area.
- May be widespread extension → Systemic HSV
- Chronic perineal ulcerative HSV → young males homosexuals in AIDS

Generalized acute mucocutaneous HSV

- Starts with localized vesicular eruption located in genital areas (in HSV2) or other areas (in HSV1)
- Rapid dissemination with Fever may occur suggestive

Systemic

- Usually follows oral or genital lesions or As
- Area of Necrosis in adrenal - pancreatic

Pathology:

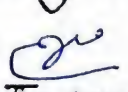
- 1) Marked intracellular edema which produces balloon cell degeneration.
- 2) Formation of thick walled vesicles by combination of intra-and inter-cellular edema.
- 3) Multinucleated giant cells in epidermis, these are epidermal cells with large number of nuclei ranging between 2 and 15.

C/P:

1) **primary infection:** differ according to site of infection:

Herpetic gingivostomatitis	Inoculation herpes simplex	keratoconjunctivitis	Herpes genitals
Begin with fever, malaise, excessive dripping, restlessness. Vesicles appear on (tounge, pharynx, palate, buccal mucosa). Vesicles present as white area surrounded by red areolas → rupture rapidly. Regional lymph nodes → enlarged, tender. Within 3-5 days → fever subside, complete recovery occur in about 2wks.	<ul style="list-style-type: none">○ Direct inoculation of skin → eruption of large bullae or scattered vesicles with enlargement and tender of lymph node. 2ry infection may occur → pustules.○ Sites:<ul style="list-style-type: none">- Finger tips (herpetic whitlow), seen in nurses and dentists.- Face and upper trunk: multiple crops of vesicles and pustules on erythematous base.	<ul style="list-style-type: none">○ 1ry herpes infection of eye, usually cause severe purulent conjunctivitis with opacity and superficial ulceration of cornea.○ Pre-auricular lymph node → enlarged, tender.	<ul style="list-style-type: none">○ 1ry infect. with type II herpes hominis virus.○ Vesicles with erosions on glans, prepuce, shaft in male and on external genitalia in females

⑩ Herpes gladiatorum: → HSV primary infection, primarily noted in wrestlers الحصاريكيب ③
→ involving extra-mucosal sites typically over face, neck, or arms.

⑪ Neonatal Herpes Simplex:  antepartum → transplacental
intrapartum → II. genitalis during delivery
postpartum → not maternal

** The risk of Transmission to the neonates from an infected mother is
(high) 50% → among women with 1st episode genital herpes near the time of delivery
and (low) <3% → among women with recurrent herpes.

Ⓐ Early intra-uterine infection: → Disturbed embryogenesis - abortion - Cong anomalies
not compatible with life

Ⓑ Late intra-uterine infection: → growth & psychomotor retardation.
Microcephaly

Ⓒ Neonatal infection: → widespread recurrent vesicular eruption (resemble epidermoly)
localized cutaneous HS in scalp or buttocks.

⑫ Genital Herpes (Herpes progenitalis): HSV 2

Primary genital Infection

2d-2w

* More Severe & prolonged than recurrent infection.

① Constitutional Symptoms

‡ painful grouped vesicles in genitalia → pustules
→ crusting → tender ulcers

② painful lymphadenopathy

Cervicitis / urethritis / proctitis.

painful erosive balanitis

Recurrent genital Herpes

* More frequent with HSV-2 > HSV-1

* Mild

① limited number of vesicles re on the genitalia or buttocks (especially in women).

② Prodrome followed by: grouped vesicles → pustule - ulceration → resolution

③ Complications are uncommon

④ Frequency of recurrence & with the severity of the first

vesicles become
ulcerative or pustular in massive crops, heal with scar. TTT:
Acyclovair in dose 5-10mg per kg, IV every 8hrs, systemic antibiotics in
case of 2ry infection.

A → aseptic meningitis
neonatal infection

2ry infection

2) Recurrent infection:

Differs from 1ry infection in:

- Smaller size of vesicles.
- Close appearance.
- Absence of constitutional symptoms.
- Examples:
 - Herpes labialis or herpes facialis: start with burning or itching sensation followed after few hrs by appearance of closely grouped vesicles on erythematous base; heal in 7-10 days without scar. Recurrence usually occurs in the same region.
 - Recurrent HS of thoracic or lumbar region: the vesicles may be arranged in line or zosteriform distribution and may be associated with deep pain and lymphadenopathy so it might be confused with HZ.

Complications of HS:

- 2ry leucoderma.
- EM with typical iris pattern may follow each attack of HS.
- Recurrent infection of eye may produce dendritic ulcer.
- Herpes genitalis in women may be associated with high incidence of cancer cervix.
- Encephalitis may complicate disseminated infection in immune deficiency disorders, malnutrition.
- kaposi's varicelliform eruption (eczema herpeticum): may complicate both 1ry and recurrent infection in certain patients include atopic patients or others as Darrier's, pemphigus foliaceus, ichthyosiform erythroderma or other inflammatory diseases. C/P: the vesicles become haemorrhagic or pustular in massive crops, heal with scar. TTT: Acyclovair in dose 5-10mg per kg, IV every 8hrs, systemic antibiotics in case of 2ry infection.

A → aseptic meningitis
B → Bacterial infection
C → Cancer cervix, scarring.
D → Depressive, Dissemination
← E → EM
N → Neonatal

2ry infection
aseptic meningitis
urine retention
Cancer Cervix
depressive -
neonatal

55745

- ③ Male & Female with st. progonitis:-

- Ovid intercourse :- there is a history of ST. progenesis in male or female.
- Spermicidal foam for ♀ and condom for ♂ followed by wash with water & soap after intercourse).
- ④ Specific vaccines to prevent recurrence
- ⑤ Non-specific immune therapy eg interferons.

1. Acyclovir 400mg
 1. Valacyclovir 1g
 1. First episode

BENEFITS OF early (H) of H. progestationalis :-

- 1) Reduce Pain & Swelling
- 2) Reduce the duration of viral shedding
- 3) Reduce the time to heal for 1st episode
- 4) Reduce Recurrent genital herpes.

H of cydovir - reactivat
herpes simplex virus
infection

Neonatal JHS

- $\frac{20 \text{ mg/kg}}{\text{IU}} / 8 \text{ hs}$ for 21 days

③ Photoactivation

Pointing the design with dye using a neutral red fading in blue. Then exposing it to fluorescent light.

* Immunocompromised

Recommended until all HSCAULT
 - Acyclovir 400 mg
 - Valacyclovir 1gm
 - Acyclovir 800 mg

(Kopas's varicilliform eruption)

Recurrence

1st 400 mg
2nd 500 mg

of all infection

- Azidothymidine 400 mg
- Valacyclovir 500 mg

Recommended until all
Mucocutaneous lesions
are healed.

* Systemic Steroids

Stipico pro genitalio

5. - Aglaia 5% + Hydrocortisone 1% cream
+ 1% 2% 3% 4% 5% 6% 7% 8% 9% 10%
(10%) 1% 2% 3% 4% 5% 6% 7% 8% 9% 10%
1% 2% 3% 4% 5% 6% 7% 8% 9% 10%

Crusted stage: antiseptic ointment + systemic antibiotic (ery infection)

TTT:

1) Systemic ttt:

a) Acyclovair:

(5-10mg/kg, IV every 8hrs, continued for 7-10days) indicated in:

- Severe neonatal herpes simplex infection.
- Disseminated HS with encephalitis.
- HS with immunodeficiency.
- Kaposi's varicilliform eruption.

Oral doses of acyclovair: (200mg 5 times/day for 5days) in less severe cases.

b) Isoprinosine: 50mg/kg.

2) Topical ttt:

a) Topical acyclovair cream, 5times daily for 5 days.

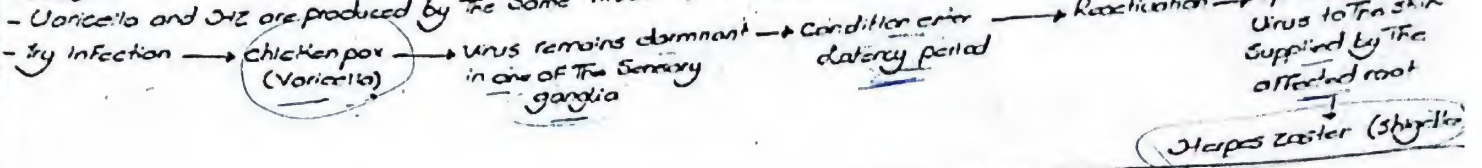
b) Zinc sulphate 4% in water may help.

c) Mild cases need no ttt.

Varicella and Herpes zoster

Etiology

- Varicella and HZ are produced by the same virus (Varicella Zoster Virus).



* Varicella (Chicken pox) *

- * Commonly affect children
- * Mode of transmission: ① Airborne droplets (usual route) ② Direct contact with vesicular fluid.
- * Incubation period: 11-20 days
- * Varicella is highly contagious → 80-90% of household contacts → develop clinical infection. → The affected individual is infectious from 1-2 days before skin lesions appear until all of vesicles have crusted.
- * Mechanism
- * During varicella infection:
 - Viral replication within regional LNs → 2-4 days → primary viraemia → viral replication in the liver, spleen, other organs → Secondary viraemia → seeds entire body (14 days post-exposure) أسبوعين
- * During this period, the virus travel to the epidermis by invading the capillary endothelial cells → then VZV travels from the mucocutaneous lesions to dorsal root ganglion cells where it remains latent until reactivation.

- * Clinically:
 - Fever → rash develops (macule → papule → vesicle (1-3mm diameter) with a clear serous fluid surrounded by a narrow red halo → pustule → crust → dew drops on a rose petal → pustule → crust → separates without scar unless secondary infected!
 - New lesions continue to develop → so different stages of lesions can be observed in the same time.
 - (polymorphic), The lesions are more numerous on trunk.
- * A person with zoster → can infect another with chicken pox → if the susceptible individual comes into direct contact with vesicular fluid.
- * Individuals with chicken pox or zoster → cannot directly give another person zoster → because herpes zoster is caused by reactivation of latent VZV.

Complications

1. try Varicella pneumonia: (adults with varicella), 10% mortality rate
2. Reye's Syndrome: Varicella + fatal encephalopathy
3. Neonatal Varicella: Fatal, Disembodied, → if the mother has developed varicella 5 days before delivery
4. Varicella in Compromised host: Dissemination to various organs e.g. varicella pneumonia → Death.

Treatment of Varicella

- ① Calamine Lotion
- ② Anti-histamine
- ③ Topical or systemic antibiotic in any infection
- ④ Anti-viral.
- ⑤ Cut nail short and wearing gloves

- distinctive feature

Varicella (chicken pox)

Def: It's the 1^{ry} infection of HZ.

C/O: Caused by varicella zoster virus.

Mode of transmission: Mainly transmitted by droplet infection, the vesicular fluid is of little importance in transmission and the dry scabs left are non infectious.

C/P:

- IP of 14-17 days.
- The disease start with fever, malaise which continue for 2 days → papular eruption → rapidly transform to vesicles, the content of vesicles may become turbid and change to pustule.
- The vesicles appear in 3-5 crops over 2-3 days.
- The predilection site: face, trunk, scalp and limbs, oral mucosa is affected in form of vesicles seen on the palate.
- Characteristic feature of the vesicles is their presence at different stage of evolution: within 2-4 days → the vesicles dry → scabs → soon separate → slight erythematous normal skin. Pruritis may be present.

N.B: Haemorrhagic Varicella: consist of high fever, severe constitutional symptoms and hgc vesicles, usually seen in: malnutrition, diseases treated by steroids, state of immunosuppression as Hodgkin's.

Complication:

- 2^{ry} infection.
- cutaneous gangrene may follow 2^{ry} infection.
- Fetal damage in form of scarring deformity and eye damage if varicella affects pregnant women.

Diagnosis:

Distinctive features of varicella are:

- 1) Centripetal distribution.
- 2) Polymorphism in each affected site.
- 3) Rapid progression to crust.

Prevention:

- 1) Prophylaxis by live attenuated vaccine.
- 2) Specific immunoglobulins is given within 4 days of contact reduce the severity but don't prevent it, it's given for neonates whose mothers has varicella 4days before or 2days after delivery and given for immunocompromised patients.

TTT:

- 1) Varicella only requires symptomatic ttt.
- 2) Acyclovair given in severe cases and as early as possible (1st or 2nd day), the doses: 10mg/kg IV every 8hrs for 5-10 days.

Herpes zoster

elderly > 50 years 5% in children

Def:

HZ is the result of reactivation of latent residual varicella virus in the sensory neurons. The precipitation factors may be: cytotoxic drugs, external trauma, radiotherapy and HZ is frequent in patients on immunosuppressive drugs or having malignancies as Hodgkin's or leukemia.

C/P:

- HZ starts by ^{1st} pain which may be severe and associated with fever, malaise, headache and tenderness localized to area supplied by one or more dorsal roots.
- After 3-4 days: appear closely set papules → rapidly become vesicles → pustules in continuous or interrupted band.
- LN in the area enlarged and tender.
- Recovery if no complication complete within 2-3wks in children and young adults and within 3-4wks in older patients.

Complication:

- 2ry infection is common in form of pustules, ulceration and may leave scar.
- Ocular complication, occur with ophthalmic zoster in 50% of cases in form of keratitis.
- Facial palsy (Ramsey-Hunt \$) in case of involvement of geniculate ganglion which may be permanent. There is also pain in ears with vesicles in pinna and external auditory canal.
- Post/herpetic neuralgia which is the commonest.

TTT:

1) Acyclovair:

- Indicated in severe cases and should be as early as possible (1st or 2nd day).
- The dose is 10mg/kg IV every 8hs for 5-10days or oral doses in less severe cases in dose of 800mg, 5times/day, for 5days.

Herpes Zoster

- Occurs in adults (>50 years) • 5% in children.
- Clinically:
 - Pain is usually 1st manifestation. severe pain
 - 3-4 days after group of vesicles on an erythematous base → arranged along the course of the sensory nerve.
 - Strictly unilateral, lesions may be hemorrhagic or necrotic & ulcerated.
 - Regional LNs: enlarged, tender
 - Healing: within 2-3 weeks, usually with scar formation.
- Mode of Transmission: (1) Direct contact (with broken blisters) • (2) Airborne droplets.
- patients are contagious (less than varicella), Newborns are at high risk of getting chicken pox from someone who has HZ
- Complications
 - Post-herpetic neuralgia (PHN) → characterized by dysaesthetic pain that persists after the skin lesions have healed.
 - affect 10-20% of HZ patients and ++ in incidence & severity with age.
 - it is either → continuous burning pain with hyperaesthesia
 - spasmodic shooting type.
 - Secondary infection → common
 - Gangrene of skin
 - HZ ophthalmicus: virus attack the gasserian ganglion (V) → GG
 - eruption on the tip of nose, upper eyelid, forehead.
 - Conjunctiva is red, swollen with superficial or deep keratitis.
 - Lesions at tip of the nose → signal possible → since nasociliary nerve is involved → which is a branch of the ophthalmic nerve.
 - ARNIS → Anterior retinal necrosis syndrome may occur with zoster of the ophthalmic nerve.
 - Ramsay-Hunt Syndrome: affection of the geniculate ganglion (GG) → ear pain/tinnitus/deafness
 - loss of taste sensation from anterior 2/3 of tongue
 - facial palsy.
 - Motor involvement: 5% of cases (commoner in older patients & those with malignancy)
 - HZ in compromised hosts → Disseminated HZ with systemic manifestations as → pneumonia, encephalitis
 - Disseminated HZ (unusual clinical presentation) → a diagnosis
 - includes 5%:
 - persistant crusted verrucous lesions in HIV-infected patient
 - post-herpetic hyperhidrosis.
 - Disseminated cutaneous disease (70% inside outside the area of primary or adjacent dermatomes).
 - atypical HZ in HIV patient
 - more than 2 dermatomes affected
 - cross midline.
 - Systemic Manifestations: pneumonia, encephalitis, gastro-enteritis.

Diagnosis:

- Culture:
 - Don't grow on ordinary culture as HZ
 - grow on tissue culture (Furman fetal diploid kidney cell).
- Pathogenic for several laboratory animals (as in HS)
- Teach-Smear → as HZ
- CR → Highly sensitive & rapid technique
- Immunology → +ve for HZ (diagnostic) if serum has at least 4 fold increase in titre of VZV

- ②

① Analgesics

- 72

- 2) Mild cases need only symptomatic ttt in form of drying lotions and antibiotics and analgesics.
- 3) Corticosteroids given in acute stages to reduce the inflammatory changes, also in severe painful cases in dose of 40mg for 10 days, withdrawal in 3wks but it may cause dissemination of HZ if patient is immunocompromised.
- 4) Post herpetic neuralgia may respond to:
 - Amitriptyline, Carbamazepine (the two drugs together).
 - Nerve block by intralesional local anesthesia and corticosteroids.
 - Acupuncture.

Treatment of PHN

- ① Analgesics
- ② Anti-viral → Aciclovir / Valacyclovir → within 72 h from 1st vesicle. Both drugs ↓ duration & pain of PHN in pt.
- ③ Corticosteroids → Used alone or in combination with anti-viral drugs
→ ↓ inflammation → ↓ further nerve damage → ↓ chronic pain associated with it.
- ④ Anti-convulsants → Carbamazepine (Tegretol) - 600-800mg/day
→ Gabapentin (Neurontin) : 1st day : 800 mg 1 tablet
2nd day : 2 tablets
3rd day : 3 tablets
(and on → doses ranging up to 3600mg/day)
* During acute Herpes zoster : Valacyclovir + Gabapentin
Together are more effective in preventing PHN than Valacyclovir alone.
→ Pregabalin (Lyrica) - Dose : 75-150 mg twice daily
- The dose may be increased to 300mg/day within 1 week on efficacy and tolerability
- in patients with reduced renal function → adjust the dose
it is primarily eliminated by renal excretion.
- ⑤ Tricyclic anti-depressants : eg amitriptyline (Tryptizol 10-25 mg) → up ↑ 75 mg
- ⑥ Nerve block : Sympathetic & Somatic nerve blocks.
- ⑦ Nerve Surgery
- ⑧ Local treatment : → EMLA cream
→ Capsaicin → 8% capsaicin patch → recently approved by FDA for tt of PHN
↓ ↓ reduce pain up to 12 weeks after a 1 hour application

Roseola infantum

Def:

Common exanthematic fever in children less than 2 years; caused by herpes virus 6.

C/P:

- IP is 10-15 days.
- The prodromal stage consist of abrupt high fever for 3-5 days, usually not accompanied by any symptoms but may periorbital oedema and haematuria → after temperature falls, an eruption appear in form of rose pink maculopapules on neck and trunk → spread to face, trunk, limbs.
- After 1-2 days the rash fades with no scaling or pigmentation.
- Cervical or occipital LN may be enlarged.
- No specific TTT required.

Infectious mononucleosis

Def:

Virus infection caused by EB virus. The 1ry infection followed by persistence of virus in lymphoid cells.

Mode of infection: by direct contact (kissing) or droplet infection.

C/P:

- IP is 33-49 days.
- Disease start by fever which persist for 5-7 days, but may continue for 2wks, associated with sore throat and membranous tonsillitis with small petechiae at junction of soft and hard palate (pathognomonic).
- Generalised enlargement of LN and most marked in cervical LN.
- Skin eruption seen in 10-15% of cases in form of macules and maculopapules on trunk, face, limbs.
- A very characteristic feature of IM is the exacerbation of the disease with antibiotics especially Ampicillin.

Diseases caused by poxvirus

Molluscum contagiosum

Def:

Mollusc contagiosum is viral infection caused by unclassified poxvirus. Consist of translucent or creamy papules with central umbilication.

Mode of transmission: Transmitted by droplet infection.

Pathogenesis:

- The virus first enters the basal keratinocytes → viral replication and production of new DNA particles → rapid cell proliferation which occur in basal and suprabasal layer.
- This proliferation lead to broadening of the rete ridges in both vertical and horizontal direction → compressing dermal papillae so appear as fibrous septa between epidermal lobules (pearl appearance).
- Later on the cells at the base of the lesion will destroy → softening of the centre and depression of the top → typical umbilication.
- Under microscope the virus appears as large hyaline bodies (purple-red by H&E) seen among the broadened rete ridges. The largest number of molluscum bodies seen in the centre of the fully developed lesion.

C/P:

- IP is 14 days-6 months.
- Site: The predilection site is face, neck and genital areas; however any site can be involved.
- Shape: The lesion is shining, pearly white, hemispherical, umbilicated papule which may show a centre pore.
- Size: a diameter of 5-10mm can be reached in 6-12wks but larger lesions may be found.
- Course:
 - Spontaneous healing may occur in some lesion.
 - Spontaneous healing start by inflammation → suppuration → crustation → destruction of the lesion.

Molluscum Contagiosum (MC):

- Self-limited
- Epidermal
- (Pox virus) infection.

→ Characterized Clinically by → Single or Multiple, skin coloured, Umbilicated papules.

- Occurring in → children / sexually active adults (20-29 yrs)
- in HIV +ve patients → numerous mollusca usually arise on the face.
- $\sigma > \rho$

Citology → Molluscum Contagiosum virus (MCV)

- Large / double-stranded DNA virus.
- 2 types → MCV-1 (Majority of infection (90%))
- MCV-2

Transmission

- ① Skin to skin contact, clothing, on towels → possible in Steam & Sauna baths.
- ② Sexual: adults.
- ③ autoinoculation: atopic patients.

Clinically

IP → 2 wks → 6 months

Site → Face, Trunk, axilla, hands.

→ Sexual Transmission: genital / lower abdomen upper thigh.

Lesion

- it is characterized by
 - Single or Multiple
 - Small (1-5mm)
 - Skin coloured or pearly white.
 - Waxy, dome shaped
 - Papule
 - with Umbilicated Center. (which become more apparent after freezing with ethyl chloride).
 - A Curd-like substance can be expressed from its center
- Sometimes The lesions become inflammatory
- Spontaneous disappearance within 6-9 months.

MC → Flat wart

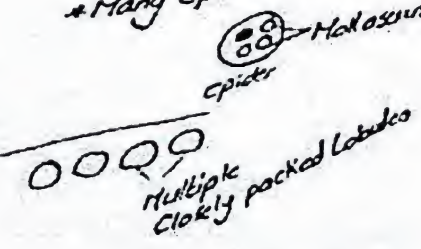
→ Syringoma.

In the patients

- ① Hundreds of lesions appear, mainly on the face
- ② Giant Molluscum may occur.
- ③ Spontaneous regression does not occur.
- ④ HAART (Highly active anti-retroviral Therapy) → lesions often resolve.

histopathology

- * The epidermis grows down into the dermis as
- * Multiple, closely packed lobules.
- * Many epidermal cells contain large intracytoplasmic inclusion bodies (Molluscum bodies) pathogen.



Treatment

- * Because the disease generally resolves spontaneously, self-limited
- * Avoid swimming pools, shared towels,
- * Curettage with or without light electro-cautery
- (Emlo cream) local anaesthesia applied 1h before therapy to pain
- in Cryotherapy
- Chemical applications of TCA or phenol to the lesion.

- Usually most cases are self limiting within 6-9 months but some lesions may persist for 3-4 years.

DD: solitary molluscum may resemble pyogenic granuloma, early keratoacanthoma, and early epithelioma. Diagnosis confirmed by pathological exam.

TTT:

- 1) Often leave to resolve spontaneously especially in young children in whom ttt will be painful or frightening.
- 2) Application of adhesive plaster every night may help to remove small lesions.
- 3) Simple mechanical method by expression of the content by squeezing the papule with blunt forceps may be successful.
- 4) Superficial curettage with sharpened wooden spatula may be sufficient.
- 5) Chemical application of caustics like silver nitrate, phenol, or strong iodine solution may be curative.
- 6) Cryotherapy may be helpful.
- 7) Diathermy should be reserved for large lesions as it leaves scars.
- 8) Topical retinoid acid has been suggested in resistant lesions.
- 9) Topical 10% properdin iodine solution and 50% salicylic acid plaster application are very effective in ttt of some lesions.

Diseases caused by Papova virus

Common infectious wart

Def:

Warts are infections of skin and contagious mucous membrane by members of papilloma virus group.

Mode of infection: warts are transmitted by direct contact or indirect by contaminated objects, or autoinoculation from hand to foot. Susceptibility to infection depends on the immune response of host.

Pathology:

- 1) Hyperkeratosis, hyperplasia of all layers of epidermis.
- 2) Rete ridges are elongated and flattened and converted towards the centre of wart.
- 3) Large vacuolated cells and clumps of keratohyalin granules within and below granular layer.
- 4) Dilated and tortuous blood vessels in dermal papillae.

Clinical features: eruption follows IP of 1-8 months (average 4 months):

1. Common wart:

- **Caused by:** HPV numbers (2, 4, 7)
- **C.P:**
 - The typical lesions are firm papules with rough horny surface.
 - Size: (few mms - over a centimetre) in diameter.
 - Site: they occur anywhere on skin but common sites are back of hands, fingers and knees. And are rarely seen in genital area.
- **DD:**
 - Large wart may like tuberculosis verrucosa but the latter surrounded by erythematous areola.
 - Acrokerato- verruciforms of Hopf seen in Darier's.
 - Subungual fibroma in tuberous sclerosis.

2. Plane wart:

- **Caused by:** HPV number 3.
- **C.P:**
 - The typical lesions are smooth flat or slight elevated papules and skin coloured or greyish white in colour.
 - The shape of lesion usually round.
 - The size is 1-5mm or more in diameter. Plane wart may coalesce.
 - Site: the sites of predilection are face, shin and back of hands.
 - Koepner phenomenon is commonly seen especially at site of scratching.
- Plane wart tends to disappear spontaneously after few wks or months but in some cases may persist for years.
- **DD:** lichen planus but LP associated with pruritis and Wickham's stria are characteristic of LP and its unusual seen on face.

3. Planter wart:

- **Caused by:** HPV number (1, 2, and 4). Mosaic wart caused by HPV 2.
- **C.P:**
 - The primary lesion is small shiny papule which soon become → sharply defined, rounded with a rough keratotic surface and surrounded by smooth ridge of thickened epidermis which not continued over surface of wart.
 - Site: the commonest sites are the pressure points, heels or the metatarsal heads.
- **Mosaic warts:** represent plaques of closely grouped warts. Planter warts are usually painful but mosaic warts are painless.
- **The duration:** is very variable, average duration before puberty is less than year while in older children and adult may persist for years.
- **DD:** a. Callosities are frequently confused with planter wart. But: Callosities have uniformly smooth surface, paring of callosity doesn't produce bleeding points and epithelial ridge around callosity continue with the lesion.

Human papilloma Virus

(9)

① Common wart (verruca vulgaris) - Firm papules, with verrucos hyperkeratotic surface

- occurring singly or in groups.
- Commonly on dorsal aspect of hands & fingers
- periungual warts are common

قال Histopathology Histopathological Criteria of Verruca Vulgaris.



- (1) Hyperkeratosis, Acanthosis, papillomatosis
- (2) Rete ridges → are elongated, point radially towards the center of the wart
- (3) groups of large vacuolated cells → in upper stratum Malpighi & in granular layer
- (4) atier of parakeratosis lies over the crest of the papillomatous elevation

Filiform wart ::

Shows a thread-like filar projection arising from a filar base.

Seen most commonly → Face / Scalp

Butchers → high incidence of VV hands.

verruca plana ::

- Slightly elevated, flat, smooth papules, linear arrangement in scratch marks (Koebner phenomenon)
- Some patients with flat warts develop clinically evident inflammation around this warts → precedes their spontaneous involution

Histopathology

- Hyperkeratosis, Acanthosis
- No papillomatosis or parakeratosis
- Numerous vacuolated cells lie in the upper stratum Malpighi & in granular cell layer
- Horny layer 'pronounced Basket-weave appearance' → resulting from vacuolation of the horny layer.

Plantar wart (v. plantaris)

- Commonly → pressure points → heel / metatarsal areas
- painful (usually)
- Usually have rough keratotic surface, studded with multiple small black dots (thrombosed capillaries within dermal papillae).
- peripheral rim of thickened skin
- When multiple warts coalesce into large plaque → it is called (Mosaic wart)

verruca profunda (Deep palmoplantar wart) ::

- Deep, tender, endophytic lesions
- They do not coalesce,
- They are usually covered with a thick callus → its removal → soft brownish tissue & small bleeding points

Histopathology in Myrmecia:



- The nuclei of epidermal cells is deeply basophilic
- Surrounded by a clear zone.
- Cytoplasm contains large, irregularly shaped, homogenous, eosinophilic (inclusions) → representing keratohyaline.

- **N.B:** Continuous paring of the planter wart result in appearance of small bleeding points, these points represent the tips of elongated dermal papillae containing dilated tortuous blood vessels.

4. Filiform and digitate wart:

Are commonly seen on face and neck, they have the surface like common wart but usually have longer stall.

5. Genital wart:

- **Caused by:** HPV 6. And sexually transmitted. *mode of infection* Sexual
non-sexual
contact
- **Microscopically:** differ from common wart in:
 - The horny layer is parakeratotic instead of hyperkeratotic.
 - The vacuolated cells in upper Malpighian layer are limited in distribution and not found in all sections. And carcinomatous changes may rarely develop.
- **C/P:** warts are soft, elongated and even pedunculated. *filiform, cauliflower*
- **Site:** The most common sites in male genitalia are glans, prepuce and shaft of penis. And in female genitalia is Vulva where masses tend to be large with bad odour. Perianal warts are similar to vulvar wart in being hyperplastic.
- **N.B:** During pregnancy, vulvar wart may attain large size which may be severe enough to obstruct labour.
- **N.B:** In genital wart, if there is sudden enlargement or signs of inflammation or prolonged duration, malignant changes should be suspected.

HPV → larger & ↑ increase risk of malignancy

Treatment:

- Prophylactic TTT:** to avoid transmission and autoinoculation:
 1. Avoid nail biting in patients having periungual warts.
 2. Planter wart should be covered by plaster or rubber socks.
 3. In genital wart, condoms should be used or abstain intercourse till healing.
 4. Towels in general should not be shared.

b. Active TTT:

1. Electrocautery:

Electrocautery is reserved for severe and persistent cases. It needs local anaesthesia as it's usually painful and it should be done carefully as it may leave scar.

2. Chemical cautery:

Salicylic acid preparation	formaldehyde	glutaraldehyde	podophyllin	other
<p>Salicylic acid can be used alone in different concentration or with other chemicals:</p> <p>a. Preparation of (Salicylic acid 16.7% and lactic acid 16.75% in flexible collodion add to 100):</p> <p>Is ttt of choice in hand and feet warts.</p> <p>Applied on wart after the surface is abraded, closed with adhesive blaster and left overnight.</p> <p>b. Using of adhesive blaster containing 40% salicylic acid: might be very useful for planter wart. It's used daily.</p>	<p>Formaldehyde (aqueous solution) is very useful in planter wart.</p> <p>Application for 15 minutes or overnight can produce cure rate up to 80% in 6-8wks of continuous use.</p>	<p>Glutaraldehyde 10% in aqueous ethanol can be used daily.</p> <p>Both glutaraldehyde, formaldehyde can cause dryness of skin and allergic contact dermatitis.</p>	<p>Used mainly for anogenital wart.</p> <p>Suitable preparation: from 15-25% in compound tincture of benzoine.</p> <p>One application may be enough or repeated until complete clearance.</p> <p>Should not be used in: pregnancy, large areas, or on bleeding surfaces.</p> <p>Podophyllin 25% in liquid paraffin may be effective in planter warts.</p>	<p>Other caustics like TCA, silver nitrate and glacial acetic acid can be used.</p>

(# of genital wart)

(11)

Therapeutic Modalities for HPV infections

- ① Chemical destruction e.g. Salicylic acid 1.5, Lactic acid 1.5
- ② Physical destruction
 - Surgical excision or electrosurgery
 - Cryotherapy
 - with Liquid nitrogen
 - less commonly → CO₂

HPVs are resistant to heat (due to absence of viral envelope)

→ especially for genital warts (11) Treatment of during pregnancy

③ Chemotherapeutic agents

- podophyllin resin 20% in collodion → For genital warts.
- podophyllotoxin 0.5% → applied by the patient at home. (twice daily) 3 days
- Sinecatechins → intraleisional → non-genital warts.
- 5FU
 - Topically
 - Vaginal & urethral warts.
 - adjuvant therapy → after laser → to ↓ recurrence

→ Laser Therapy → Followed by Topical 5FU → to ↓ recurrence

Oral retinoids → For EU → as antiproliferative antiangiogenesis.

Imiquimod 5% cream (Aldara) → used in # of Condyloma acuminata in adults.

• immune response modifier

• Unique → it does not rely on physical destruction of the lesion as other therapies. But it is directed at eradication of the causative agent (HPV)

Mechanism

- after viral infection → Keratinocytes and other cells release cytokines e.g. INF- α , TNF- α , IFN- γ is cytokine → act to protect adjacent keratinocytes from viral infection
- Until specific CMV response is induced
- Topically applied Imiquimod (Aldara) can induce keratinocytes and other cells to release INF- α → to inhibit viral replication directly & to promote stronger CMV

Application

- Cream → supplied in single dose packet.
- applied to the involved area by the patient
- 3 times / week at bedtime
- washed after 6-10 hours
- Treatment duration is up to 16 weeks

Side effects

Local irritation → erythema
itching
crusts.

حركات في اسبوع قبل البزوغ
يفضل بعد 7 ساعات
في شعور

ie immunomodifiers

- interferon α 2b → intraleisional → 3 times / week For Condyloma acuminata
- Systemic # by SC or IV route with interferon → also been tried.

otherapy of intraleisional injection of Candida antigen.

3. Cryotherapy:

Freezing of wart by CO₂ snow or liquid nitrogen is frequently used for treatment of warts. If warts don't clear from the first application further application can be applied every 2 wks.

4. Surgical method:

Excision should be avoided as it produces scarring and recurrence of warts is frequent.

5. Other methods:

- Retinoic acid: can be used in plane wart or even warts.
- Etritinate may be helpful in some difficult or wide spread of plane warts.
- 5-fluorouracil (5-FU): 5% of 5-FU is effective in treatment of plane wart as well as planter warts. It acts by inhibiting HPV replication.
- Dinitrochlorobenzene(DNCB).
- Bleomycin.
- Interferon: may be useful in treatment of severe disseminated warts in immunodeficiency patients.

✓ ... therapy → after laser → to ↓ recurrence

- ④ Laser Therapy → Followed by Topical 5FU → to ↓ recurrence
- ⑤ Oral retinoids → For EU → as antiproliferative antiangiogenesis.

⑥ Imiquimod 5% cream (Aldara) → used in # of *Candidioma acuminata* in adults.

- immune response modifier
- Unique → it does not rely on physical destruction of the lesion as other therapies. But it is directed at eradication of the causative agent (HPV)

• Mechanism

- after viral infection → Keratinocytes and other cells release cytokines e.g. $INF\alpha$, TNF , $IL-1$
- $INF-\gamma$ is cytokine → act to protect adjacent Keratinocytes from viral infection until specific CH2 response is induced
- Topically applied Imiquimod (Aldara) can induce Keratinocytes and other cells to release $INF-\alpha$ → to inhibit viral replication directly & to promote stronger CH2 response

• Application

- Cream → supplied in a single dose packet.
- applied to the involved area by the patient
- 3 times / week at bedtime
- washed after 6-10 hours
- Treatment duration is up to 16 weeks

• Side effects

Local irritation → erythema
itching
crusts.

طريقا في الصباح قبل النوم
يغسله بعد 7 ساعات
في شمس

mic immunomodifiers

- interferon α 2b → intradermally → 3 times / week For *Candidioma acuminata*
- E. clamsir # hu for AD route with interferon → also ben tied.

Epidermodysplasia verruciformis (EV)

Def:

EV is an AR hereditary disorder. In which there is a distinctive pattern of abnormal response to some HPV.

Aetiology: more than one HPV can be responsible for development of EV, examples:

- HPV3: cause flat wart-like lesions involving hands, face and whole skin except trunk.
- HPV5: cause flat wart-like, pityriasis versicolour like, depigmented and pigmented plaques. Affect whole skin including trunk.
- HPV8: cause flat wart-like red plaques, pigmented and depigmented plaques involve whole skin.
- HPV9: cause flat wart-like and red plaques involving whole skin.
- HPV10: induces flat warts and elevated flat warts involving whole skin.
- HPV14 and 15: induces flat wart-like lesions and red plaques involving whole skin.

Pathology: the histological picture is like of plane wart but with frequent vacuolated cells seen within and below granular layer.

Clinical features:

- The disease characterised by early and progressive development of plane wart-like lesions on face and neck, and common wart-like lesions which tend to be larger and firmer on the trunk and extremities. The lesion tends to be confluent in large plaques.
- The wart usually starts in early childhood but may appear at any age.
- The lesion in EV are divided into:
 - a. Benign lesions:
 - Pityriasis rosea like lesions.
 - Common wart-like lesions: on trunk, limbs.
 - Plane wart-like lesions: on face.
 - Psoriasis-like-lesions: on elbow, knee.

Epidemiology Verruiformis (EV) Q: Epidemiology Verruiformis.

- Rare. AR, Familial skin disease usually begins in childhood.

→ Clinically it is characterized by ① long lasting, widespread flat, wart-like lesions → Face/arms/legs/back of hands.
② Malar erythema M-like lesions → Face/neck / Trunk/arms → tend to become confluent

Etiology

→ Malignant transformation → of some cutaneous lesions occur in 1/2 of patients

→ Different HPV types occur in EV → frequently 588 only HPV5 (Found in most cancers), HPV8 (oncogenic potential)

→ EV types → induce EV-like eruptions → in immunosuppressed patients (e.g. AIDS, lymphoproliferative)

→ EV-related HPV → have been isolated from normal skin of healthy individuals (highly sensitive DNA detection)

→ indicating that normal population act as reservoir for EV-types.

→ Recently a mutation in 2 genes EVER1 & EVER2 have been identified as cause of EV.

Pathology

Ident on The HPV type involved.

3 → induce flat wart like lesions → similar to that of common wart.

5 (more extensive lesions) → swollen keratinocytes with light blue cytoplasm (flatoid?)

→ nuclei ranging from small & pyknotic → large with margined chromatin

b. Carcinomatous lesions:

- Bowen's disease which detected as raised plaques that may be red or pigmented. They may look like psoriasis in beginning but development of crusts and ulceration on the lesions suspect for malignant transformation.

DD: TVC, LP, Psoriasis, Bowen's carcinoma, Acrokerato- verruciformis of Hopf.

TTT:

1. The patient should avoid sun exposure and X-rays as these may accelerates development of Bowen's disease.
2. Oral retinoid as Etretinate in dose of 1mg/kg/day was found to be effective in many cases.

Bowenoid papulosis

Type of intraepithelial neoplasia

Cause → HPV

Benign course. malignant transformation in 2%

HIP → scattered atypical cell or full thickness atypia
loss of granular layer

CIP → small red/brown flesh wheeled flat or warty lesion of labia & penis

HP → regress spontaneously
if persist HP as genital wart

acrokeratoma verruciformis

genodermatoses

disorder of keratinization due to ATP2A2 gene AD
Occur in Darier disease

CIP → multiple flat-topped symmetrical, skin coloured keratotic lesion on dorsum of hand

• Common in children

• may be asymptomatic

• associated is nail dystrophy.

HIP → acanthosis - hyperkeratosis, papillomatosis
granular layer thick.
acantholysis → rare

crunch-spine → epidermal elevation

HP → cryotherapy
laser
isotretin